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NEWS 2	JUL 02	LMEDLINE coverage updated
NEWS 3	JUL 02	SCISEARCH enhanced with complete author names
NEWS 4	JUL 02	CHEMCATS accession numbers revised
NEWS 5	JUL 02	CA/CAplus enhanced with utility model patents from China
NEWS 6	JUL 16	CAplus enhanced with French and German abstracts
NEWS 7	JUL 18	CA/CAplus patent coverage enhanced
NEWS 8	JUL 26	USPATFULL/USPAT2 enhanced with IPC reclassification
NEWS 9	JUL 30	USGENE now available on STN
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NEWS 11	AUG 06	FSTA enhanced with new thesaurus edition
NEWS 12	AUG 13	CA/CAplus enhanced with additional kind codes for granted patents
NEWS 13	AUG 20	CA/CAplus enhanced with CAS indexing in pre-1907 records
NEWS 14	AUG 27	Full-text patent databases enhanced with predefined patent family display formats from INPADOCDB
NEWS 15	AUG 27	USPATOLD now available on STN
NEWS 16	AUG 28	CAS REGISTRY enhanced with additional experimental spectral property data
NEWS 17	SEP 07	STN AnaVist, Version 2.0, now available with Derwent World Patents Index
NEWS 18	SEP 13	FORIS renamed to SOFIS
NEWS 19	SEP 13	INPADOCDB enhanced with monthly SDI frequency
NEWS 20	SEP 17	CA/CAplus enhanced with printed CA page images from 1967-1998
NEWS 21	SEP 17	CAplus coverage extended to include traditional medicine patents
NEWS 22	SEP 24	EMBASE, EMBAL, and LEMBASE reloaded with enhancements
NEWS 23	OCT 02	CA/CAplus enhanced with pre-1907 records from Chemisches Zentralblatt
NEWS 24	OCT 19	BEILSTEIN updated with new compounds

NEWS EXPRESS 19 SEPTEMBER 2007: CURRENT WINDOWS VERSION IS V8.2, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 19 SEPTEMBER 2007.

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FILE 'HOME' ENTERED AT 07:40:40 ON 30 OCT 2007

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FILE 'CAPLUS' ENTERED AT 07:40:51 ON 30 OCT 2007
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FILE COVERS 1907 - 30 Oct 2007 VOL 147 ISS 19
FILE LAST UPDATED: 29 Oct 2007 (20071029/ED)

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=> s AGEs
L1 60471 AGES

=> s l1 and inhibit?
1974279 INHIBIT?
L2 3571 L1 AND INHIBIT?

=> s l2 and diabet?
149276 DIABET?
L3 573 L2 AND DIABET?

=> s l3 and py<2003
22908219 PY<2003
L4 263 L3 AND PY<2003

=> s l4 and nephropathy?
14476 NEPHROPATHY?
L5 62 L4 AND NEPHROPATHY?

=> d ibib abs hitstr 1-10

L5 ANSWER 1 OF 62 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:624272 CAPLUS
DOCUMENT NUMBER: 139:270775
TITLE: Role of dyslipidemia and AGE/ALE formation in the progression of nephropathy and retinopathy in STZ-diabetic rats
AUTHOR(S): Thorpe, Suzanne R.; Alderson, Nathan L.; Chachich,

Mark E.; Januszweski, Andrzej; Youssef, Nancy N.; Jimenez, Stephanie M.; Gardiner, Tom; Frizzell, Norma; Canning, Paul; Lichanska, Agnieszka; Baynes, John W.; Stitt, Alan W.

CORPORATE SOURCE: Graduate Science Research Center, Department of Chemistry and Biochemistry, University of South Carolina, Columbia, SC, 29208, USA

SOURCE: International Congress Series (2002), 1245(Maillard Reaction in Food Chemistry and Medical Science), 169-173
CODEN: EXMDA4; ISSN: 0531-5131
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We hypothesized that the beneficial effects of a variety of pharmacol. agents on the progression of diabetic complications were mediated by a common pathway limiting the formation of advanced glycation and advanced lipoxidn. end products (AGEs/ALEs) on protein. We studied the effects of the AGE/ALE inhibitor pyridoxamine (PM), the antioxidant vitamin E (VE) and the ACE inhibitor enalapril (EP) on the development of nephropathy and retinopathy in STZ-induced diabetic rats over 29 wk. Blood glucose and glycoHb were similar in all diabetic groups. Plasma lipids rose continuously in diabetic animals and only PM significantly attenuated this increase. Early nephropathy was indicated by increased plasma creatinine, and urinary albumin, protein and TGF-β excretion in untreated rats. While all interventions limited renal damage to some extent, PM was the most effective, although the increased expression of renal laminin β1 and fibronectin mRNA was normalized by all therapies. Measurement of retinal damage (cellular capillaries, vascular basement membrane-associated laminin) showed that only PM significantly limited signs of early retinopathy in diabetic rats. Only PM limited the increases in the AGE/ALEs in renal and retinal tissue, and in skin collagen, of diabetic rats. Our results suggest that limiting both dyslipidemia and AGE/ALE formation is required for maximum protection of renal and retinal function in the STZ-diabetic rat.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 62 CAPLUS

ACCESSION NUMBER: 2003:267440 CAPLUS
DOCUMENT NUMBER: 139:131842

TITLE: Effect of advanced glycosylation end products on matrix metalloproteinase-2 activity in rat renal cortex

AUTHOR(S): Yu, Xiaoyan; Li, Cai; Miao, Chunsheng; Zhang, Xiuyun
CORPORATE SOURCE: Institute of Biological Engineering, Jilin University, Changchun, 130021, Peop. Rep. China

SOURCE: Jilin Daxue Xuebao, Yixueban (2002), 28(4), 331-334
CODEN: JDXYA3; ISSN: 1671-587X
PUBLISHER: Baiqiuen Yike Daxue Xuebao Bianjibu
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB The effects of advanced glycosylation end products (AGEs) and aminoguanidine (AG) on matrix metalloproteinase-2 (MMP-2) activity in rat renal cortex were studied. Normal rats were given by tail vein injection with either AGE-modified rat serum protein (AGE-RSP), AGE-RSP followed by i.p. injection of AG (AGEs + AG), or native rat serum protein (native RSP), while normal rats without any treatment were as a control. AGEs amount in renal cortex was quantified by ELISA and MMP-2 activity was examined by zymog. anal. AGEs in renal cortex was

markedly increased and the activity of active form MMP-2 (62 KD) was decreased significantly in rats treated with AGE-RSP for 4 wk as compared with those in rats treated with native RSP or in control. AGEs decreased the activity of MMP-2 in renal cortex and AG inhibited the effect of AGEs on the activity of MMP-2, suggesting that AGEs may induce a decrease in degradation of ECM through inhibition of the MMP-2 activity and result in ECM accumulation.

L5 ANSWER 3 OF 62 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:88355 CAPLUS
DOCUMENT NUMBER: 139:90216
TITLE: Studies on the processing of herbal medicines (III): HPLC analysis of magnolol and inhibitory effects on the formation of advanced glycation end products (AGEs) in vitro of unprocessed and processed Magnolia Bark
AUTHOR(S): Kim, Jin Sook; Kim, Hyeun Jeong; Ko, Jin Hee
CORPORATE SOURCE: Department of Herbal Pharmaceutical Development, Korea Institute of Oriental Medicine, Seoul, 135-100, S. Korea
SOURCE: Saengyak Hakhoechi (2002), 33(4), 308-311
CODEN: SYHJAM; ISSN: 0253-3073
PUBLISHER: Korean Society of Pharmacognosy
DOCUMENT TYPE: Journal
LANGUAGE: Korean
AB Advanced glycation end products (AGEs) are largely involved in the pathogenesis of diabetic nephropathy. It is obvious that inhibition of AGEs formation is important in preventing the occurrence and progression of diabetic nephropathy. In diabetes, this reaction is greatly accelerated and is important in the pathogenesis of diabetic complications, especially diabetic nephropathy. Therefore, to seek possible AGEs inhibitors in herbal medicines, unprocessed - and processed Magnolia Bark were examined in vitro as basic data for animal experiment The content of magnolol in unprocessed Magnolia Bark was 0.796±0.072%, and after processing was decreased to 0.586±0.101 % ($p<0.01$). The content of AGEs was measured by their intrinsic fluorescence. The IC50 (μg/mL) values of aminoguanidine, unprocessed - and processed Magnolia Bark are 38.845± 8.36 μg/mL, 54.264±3.153 μg/mL and 27.882±1.836 μg/mL, resp. This result means that processed Magnolia Bark was more effective than aminoguanidine, as pos. control.

L5 ANSWER 4 OF 62 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:939497 CAPLUS
DOCUMENT NUMBER: 138:151440
TITLE: Activation of tubular epithelial cells in diabetic nephropathy
AUTHOR(S): Morcos, Michael; Sayed, Ahmed A. R.; Bierhaus, Angelika; Yard, Benito; Waldherr, Rudiger; Merz, Wolfgang; Kloeting, Ingrid; Schleicher, Erwin; Mentz, Stefani; El Baki, Randa F. Abd; Tritschler, Hans; Kasper, Michael; Schwenger, Vedat; Hamann, Andreas; Dugi, Klaus A.; Schmidt, Anne-Marie; Stern, David; Ziegler, Reinhard; Haering, Hans U.; Andrassy, Martin; Van der Woude, Fokko; Nawroth, Peter P.
CORPORATE SOURCE: Department of Internal Medicine 1 and Department of Nephrology, University of Heidelberg, Heidelberg, 69115, Germany
SOURCE: Diabetes (2002), 51(12), 3532-3544
CODEN: DIAEAZ; ISSN: 0012-1797
PUBLISHER: American Diabetes Association
DOCUMENT TYPE: Journal

LANGUAGE: English

AB Previous studies have shown that renal function in type 2 diabetes correlates better with tubular changes than with glomerular pathol. Since advanced glycation end products (AGEs; AGE-albumin) and in particular carboxymethyllysine (CML) are known to play a central role in diabetic nephropathy, we studied the activation of nuclear factor κB (NF-κB) in tubular epithelial cells in vivo and in vitro by AGE-albumin and CML. Urine samples from healthy control subjects (n = 50) and type 2 diabetic patients (n = 100) were collected and tested for excretion of CML and the presence of proximal tubular epithelial cells (pTECs). CML excretion was significantly higher in diabetic patients than in healthy control subjects ($P < 0.0001$) and correlated with the degree of albuminuria ($r = 0.7$, $P < 0.0001$), while there was no correlation between CML excretion and HbA1c ($r = 0.03$, $P = 0.76$). Urine sediments from 20 of 100 patients contained pTECs, evidenced by cytokeratin 18 positivity, while healthy control subjects (n = 50) showed none ($P < 0.0001$). Activated NF-κB could be detected in the nuclear region of excreted pTECs in 8 of 20 patients with pTECs in the urine sediment (40%). Five of eight NF-κBp65 antigen-pos. cells stained pos. for interleukin-6 (IL-6) antigen (62%), while only one of the NF-κB-neg. cells showed IL-6 positivity. PTECs in the urine sediment correlated pos. with albuminuria ($r = 0.57$, $P < 0.0001$) and CML excretion ($r = 0.55$, $P < 0.0001$). Immunohistochem. in diabetic rat kidneys and a human diabetic kidney confirmed strong expression of NF-κB in tubular cells. To further prove an AGE/CML-induced NF-κB activation in pTECs, NF-κB activation was studied in cultured human pTECs by electrophoretic mobility shift assays (EMSAs) and Western blot. Stimulation of NF-κB binding activity was dose dependent and was one-half maximal at 250 nmol/l AGE-albumin or CML and time dependent at a maximum of activation after 4 days. Functional relevance of the observed NF-κB activation was demonstrated in pTECs transfected with a NF-κB-driven luciferase reporter plasmid and was associated with an increased release of IL-6 into the supernatant. The AGE- and CML-dependent activation of NF-κBp65 and NF-κB-dependent IL-6 expression could be inhibited using the soluble form of the receptor for AGEs (RAGE) (soluble RAGE [sRAGE]), RAGE-specific antibody, or the antioxidant thioctic acid. In addition transcriptional activity and IL-6 release from transfected cells could be inhibited by overexpression of the NF-κB-specific inhibitor κBα. The findings that excreted pTECs demonstrate activated NF-κB and IL-6 antigen and that AGE-albumin and CML lead to a perpetuated activation of NF-κB in vitro infer that a perpetuated increase in proinflammatory gene products, such as IL-6, plays a role in damaging the renal tubule.

REFERENCE COUNT: 127 THERE ARE 127 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 62 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:923674 CAPLUS

DOCUMENT NUMBER: 138:383327

TITLE: Renal connective tissue growth factor induction in experimental diabetes is prevented by aminoguanidine

AUTHOR(S): Twigg, Stephen M.; Cao, Zemin; McLennan, Sue V.; Burns, Wendy C.; Brammar, Gail; Forbes, Josephine M.; Cooper, Mark E.

CORPORATE SOURCE: Royal North Shore Hospital, Kolling Institute of Medical Research, University of Sydney, St. Leonards, 2065, Australia

SOURCE: Endocrinology (2002), 143(12), 4907-4915
CODEN: ENDOAO; ISSN: 0013-7227

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal
LANGUAGE: English
AB The aim of this study was to determine whether aminoguanidine (AG), an inhibitor of advanced glycation, prevents expression of the profibrotic cytokine, connective tissue growth factor (CTGF), as well as accumulation of the previously reported CTGF-dependent matrix protein, fibronectin, in a model of exptl. diabetic nephropathy . Diabetic animals were randomly allocated into groups receiving 32 wk of AG or vehicle. Diabetic rats showed increases in CTGF mRNA and protein expression as well as in advanced glycation end-product (AGE) and fibronectin immunostaining, compared with nondiabetic rats. In the diabetic kidney, the increase in CTGF gene and protein expression as well as expression of the extracellular matrix protein fibronectin were prevented by AG. To further explore the relationship between AGEs and mesangial CTGF and fibronectin production, cultured human mesangial cells were exposed in vitro to soluble AGE-BSA and carboxymethyl lysine-BSA, and this led to induction of both CTGF and fibronectin. On the basis of our in vitro findings in mesangial cells linking AGEs to CTGF expression, the known prosclerotic effects of CTGF, and the ability of AG to attenuate mesangial expansion, it is postulated that the antifibrotic effects of AG in this animal model may be partially mediated by CTGF.
REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 6 OF 62 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:862270 CAPLUS
DOCUMENT NUMBER: 138:331512
TITLE: ALT-946 and aminoguanidine, inhibitors of advanced glycation, improve severe nephropathy in the diabetic transgenic (mREN-2)27 rat
AUTHOR(S): Wilkinson-Berka, Jennifer L.; Kelly, Darren J.; Koerner, Suzanne M.; Jaworski, Kassie; Davis, Belinda; Thallas, Vicki; Cooper, Mark E.
CORPORATE SOURCE: Department of Physiology, University of Melbourne, Parkville, Australia
SOURCE: Diabetes (2002), 51(11), 3283-3289
CODEN: DIAEAZ; ISSN: 0012-1797
PUBLISHER: American Diabetes Association
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The severe diabetic nephropathy that develops in the hypertensive transgenic (mRen-2)27 rat with streptozotocin (STZ) diabetes has previously been considered angiotensin II-dependent. Because metabolic pathways are also activated in the diabetic kidney, the present study aimed to determine whether renoprotection could be afforded with inhibitors of advanced glycation end products (AGEs), ALT-946, and aminoguanidine (AG). At 6 wk of age, nondiabetic control and STZ diabetic Ren-2 rats were randomized to receive vehicle, ALT-946 (1 g/l), or AG (1 g/l) and were studied for 12 wk. Systolic blood pressure was unchanged with diabetes, ALT-946, or AG. Both kidney weight and glomerular filtration rate were increased with diabetes and unchanged with ALT-946 or AG. ALT-946 and AG equally ameliorated glomerulosclerosis and medullary pathol.; however, ALT-946 did reduce cortical tubular degeneration to a greater extent than AG. Albumin excretion rate, which was elevated with diabetes, was reduced with ALT-946 but not AG. AGE immunolabeling was increased in glomeruli and reduced with ALT-946 and AG. These findings indicate that even in the context of renal injury presumed to be primarily blood pressure-and/or angiotensin II-dependent, approaches that interfere with metabolic pathways such as inhibitors of AGE formation can confer renal protection in exptl. diabetes.
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 62 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:862269 CAPLUS
DOCUMENT NUMBER: 138:104925
TITLE: Reduction of the accumulation of advanced glycation end products by ACE inhibition in experimental diabetic nephropathy
AUTHOR(S): Forbes, Josephine M.; Cooper, Mark E.; Thallas, Vicki; Burns, Wendy C.; Thomas, Merlin C.; Brammar, Gail C.; Lee, Fiona; Grant, Sharon L.; Burrell, Louise A.; Jerums, George; Osicka, Tanya M.
CORPORATE SOURCE: Department of Medicine, University of Melbourne, Austin, Australia
SOURCE: Diabetes (2002), 51(11), 3274-3282
CODEN: DIAEAZ; ISSN: 0012-1797
PUBLISHER: American Diabetes Association
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effect of ACE inhibition on the formation of advanced glycation end products (AGEs) and oxidative stress was explored. Streptozocin-induced diabetic animals were randomized to no treatment, the ACE inhibitor ramipril (3 mg/L), or the AGE formation inhibitor aminoguanidine (1 g/L) and followed for 12 wk. Control groups were followed concurrently. Renal AGE accumulation, as determined by immunohistochem. and both serum and renal fluorescence, were increased in diabetic animals. This was attenuated by both ramipril and aminoguanidine to a similar degree. Nitrotyrosine, a marker of protein oxidation, also followed a similar pattern. The receptor for AGEs, gene expression of the membrane-bound NADPH oxidase subunit gp91phox, and nuclear transcription factor-κB were all increased by diabetes but remained unaffected by either treatment regimen. Two other AGE receptors, AGE R2 and AGE R3, remained unchanged for the duration of the study. The present study has identified a relationship between the renin-angiotensin system and the accumulation of AGEs in exptl. diabetic nephropathy that may be linked through oxidative stress.
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 62 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:699556 CAPLUS
DOCUMENT NUMBER: 138:36717
TITLE: Advanced glycation end products and diabetic complications
AUTHOR(S): Jenkins, Alicia J.; Cooper, Mark E.; Stitt, Alan W.
CORPORATE SOURCE: Dept of Ophthalmology, Royal Victoria Hospital, Queen's University of Belfast, UK
SOURCE: Expert Opinion on Investigational Drugs (2002), 11(9), 1205-1223
CODEN: EOIDER; ISSN: 1354-3784
PUBLISHER: Ashley Publications Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Diabetic complications are major cause of morbidity and mortality in patients with diabetes. While the precise pathogenic mechanism(s) underlying conditions such as diabetic retinopathy, diabetic nephropathy and increased risk of atherosclerosis remain ill-defined, it is clear that hyperglycemia is a primary factor that initiates and promotes complications. Formation of advanced glycation end products (AGEs) correlate with glycemic control, and these reactive adducts form on DNA, lipids and proteins where they represent pathophysiol. modifications that precipitate dysfunction at a

cellular and mol. level. Many of these adducts form rapidly during diabetes and promote progression of a raft of diabetes -related complications. Recent evidence also suggests an important interaction with other pathogenic mechanisms activated within the diabetic milieu. This review outlines the nature of AGE formation in biol. systems and highlights accumulative evidence that implicates these adducts in diabetic complications. As more therapeutic agents are developed to inhibit AGE formation or limit their pathogenic influence during chronic diabetes, it is becoming clear that these anti-AGE strategies have an important role to play in the treatment of diabetic patients.

REFERENCE COUNT: 189 THERE ARE 189 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 62 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:695943 CAPLUS
DOCUMENT NUMBER: 137:216780
TITLE: Preparation of aromatic carboxamides as modulators of receptor for advanced glycated end products (RAGE).
INVENTOR(S): Mjalli, Adnan M. M.; Andrews, Rob; Wysong, Christopher
PATENT ASSIGNEE(S): Transtech Pharma, Inc., USA
SOURCE: PCT Int. Appl., 95 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2002070473	A2	20020912	WO 2002-US6707		20020305 <--
WO 2002070473	A3	20021227			
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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
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AU 2002245591	A1	20020919	AU 2002-245591		20020305 <--
AU 2002245591	B2	20070517			
US 2002193432	A1	20021219	US 2002-91759		20020305 <--
EP 1377295	A2	20040107	EP 2002-713758		20020305
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
CN 1494425	A	20040505	CN 2002-805994		20020305
JP 2005500254	T	20050106	JP 2002-569794		20020305
AU 2007203289	A1	20070802	AU 2007-203289		20070717
PRIORITY APPLN. INFO.:			US 2001-273403P	P	20010305
			US 2001-273404P	P	20010305
			US 2001-273429P	P	20010305
			US 2001-273445P	P	20010305
			US 2001-273446P	P	20010305
			US 2001-273454P	P	20010305
			US 2001-273455P	P	20010305
			AU 2002-245591	A3	20020305
			WO 2002-US6707	W	20020305

OTHER SOURCE(S): MARPAT 137:216780
AB G2R1R2CG1CONR3R4 [I; G1 = alkylene; G2 = H, alkyl, aryl, alkylaryl, amino,

(substituted) imidazolyl; R1 = H, alkyl, aryl, alkylaryl; R2 = alkyl, aryl, aralkyl, etc.; R3 = H, alkyl, alkylaryl, alkoxyaryl; R4 = alkylaryl, alkoxyaryl, aryl], were prepared I are modulators of the interaction between the receptor for advanced glycated end products (RAGE) and its ligands, such as advanced glycated end products (AGEs), S100/calgranulin/EN-RAGE, β-amyloid and amphoterin. I are useful in treating inflammation, the development of diabetic late complications such as increased vascular permeability, nephropathy , atherosclerosis, and retinopathy, the development of Alzheimer's disease, erectile dysfunction, and tumor invasion and metastasis. Thus, 3-(3-tert-butoxyphenyl)-3-(9-fluorenylmethoxycarbonylamino)propionic acid, HTBU, diisopropylethylamine, and 2,4-bis-(3-diethylaminopropoxy)aniline (preparation given) were stirred overnight in MeCN to give 3-(3-tert-butoxyphenyl)-3-(9-fluorenylmethoxycarbonylamino)propionic acid 2,4-bis-(3-diethylaminopropoxy)aniline amide. The latter showed IC50<0.5 μM for inhibition of binding of RAGE to s100b.

L5 ANSWER 10 OF 62 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:550257 CAPLUS
DOCUMENT NUMBER: 138:53325
TITLE: Diabetic nephropathy and carbonyl stress
AUTHOR(S): Miyata, Toshio; Yoshino, Atsushi; Kurokawa, Kiyoshi
CORPORATE SOURCE: Department of Internal Medicine, Tokai University School of Medicine, Japan
SOURCE: Furi Rajikaru no Rinsho (2000), 15, 44-49
CODEN: FRRIFI
PUBLISHER: Nihon Igakukan
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review. Research on the role of advanced glycation end products (AGEs) in diabetes has brought new insights to non-enzymic biochem. of carbohydrates and lipids. It has revealed the increased progressive, irreversible protein modifications with reactive carbonyl compds. (RCOs), derived from oxidative and non-oxidative chemical, in diabetic glomerular lesions ("carbonyl stress"). Under carbonyl stress, not only AGEs derived from carbohydrates but also advanced lipoxidn. end products (ALEs) derived from lipids accumulate in expanded mesangial matrix and nodular lesions. Carbonyl stress might be derived from hyperglycemia (lipemia), oxidative stress, and/or impaired detoxification of RCOs such as by the enhanced polyol pathway. It remains unknown whether carbonyl stress merely represents long-term accumulation of protein modifications and is inert surrogate marker or, alternatively, whether it plays an active role in the pathogenesis. Recent studies however support the latter hypothesis. Proteins modified with AGEs/ALEs initiate a range of cellular responses. Intermediates RCOs per se induce the structure and functional alterations of matrix and cellular proteins. Carbonyl stress indeed mediates the intracellular signaling by multiple pathways. The manipulation of carbonyl stress may therefore open new therapeutic approaches. Among them are the acceleration of detoxification of RCOs and the entrapment of RCOs chemical These might be achieved in various ways. These therapeutic approaches indeed inhibit the carbonyl modification of proteins with carbohydrates and lipids, prevent the intracellular protein-tyrosine phosphorylation and cellular responses, and effectively retard the development of tissue damage in vivo in animal models.

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L2          3571 S L1 AND INHIBIT?
L3           573 S L2 AND DIABET?
L4           263 S L3 AND PY<2003
L5            62 S L4 AND NEPHROPATHY?

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FULL ESTIMATED COST                                   1.14      41.11

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)        SINCE FILE      TOTAL
                                                     ENTRY    SESSION
CA SUBSCRIBER PRICE                                   0.00      -7.80

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